Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
11	9051	catalase\$1	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
L2	2967	alcaligenes or deleya or aquamarinus or microscilla or furvescens	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
(E)	47	1 same 2	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
(L4)	77	1 near5 (muta\$10 or variant\$1)	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:16

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
LI	9051	catalase\$1	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
L2	2967	alcaligenes or deleya or aquamarinus or microscilla or furvescens	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
(3)	47	1 same 2	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
L4	77	1 near5 (muta\$10 or variant\$1)	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:16

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040005655 A1

TITI F

Catalases

PUBLICATION-DATE:

January 8, 2004

INVENTOR-INFORMATION:

NAME

STATE COUNTRY RULE-47

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Voorhees

NJ US

APPL-NO:

09/922185

DATE FILED: August 2, 2001

RELATED-US-APPL-DATA:

child 09922185 A1 20010802

parent division-of 09412347 19991005 US GRANTED

parent-patent 6410290 US

child 09412347 19991005 US

parent continuation-of 08951844 19971016 US GRANTED

parent-patent 6074860 US

child 08951844 19971016 US

parent division-of 08674887 19960703 US GRANTED

parent-patent 5939300 US

US-CL-CURRENT: 435/69.1, 435/136, 435/320.1, 435/325, 536/23.2

ABSTRACT:

Catalase enzymes derived from bacterial from the genera Alcaligenese (Deleva) and Microscilla are disclosed. The enzymes are produced from native or recombinant host cells and can be utilized to destroy or detect hydrogen peroxide, e.g., in production of glyoxylic acid and in glucose sensors, and in processes where hydrogen peroxide is used as a bleaching or antibacterial agent, e.g. in contact lens cleaning, in bleaching steps in pulp and paper preparation and in the pasteurization of dairy products.

	KWIC	
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Abstract Paragraph - ABTX (1):

Catalase enzymes derived from bacterial from the genera Alcaligenese (Deleva) and Microscilla are disclosed. The enzymes are produced from native or recombinant host cells and can be utilized to destroy or detect hydrogen peroxide, e.g., in production of glyoxylic acid and in glucose sensors, and in processes where hydrogen peroxide is used as a bleaching or antibacterial agent, e.g. in contact lens cleaning, in bleaching steps in pulp and paper preparation and in the pasteurization of dairy products.

Brief Description of Drawings Paragraph - DRTX

[0012] FIG. 1 shows the full-length DNA sequence and the corresponding deduced amino acid sequence for <u>Alcaligenes (Deleya) aquamarinus</u> Catalase-64CA2.

Brief Description of Drawings Paragraph - DRTX (4):

[0013] FIG. 2 shows the full-length DNA sequence and the corresponding deduced amino acid sequence for <u>Microscilla</u> furrvescens <u>Catalase</u> 53CA1.

Detail Description Paragraph - DETX (20):

[0032] With respect to <u>Alcaligenes</u> (Delaya) <u>aquamarinus</u>, the protein with the closest amino acid sequence identity of which the inventors are currently aware is the <u>Microscilla furvescens catalase</u> (59.5% protein identity; 60% DNA identity). The next closest is a Mycobacterium tuberculosis <u>catalase</u> (KatG), with a 54% protein identity.

Detail Description Paragraph - DETX (21):

[0033] With respect to <u>Microscilla furvescens</u>, the protein with the closest amino acid sequence identity of which the inventors are currently aware is <u>catalase</u> I of Bacillus stearothermophilus, which has a 69% amino acid identity.

Detail Description Paragraph - DETX (75):

[0085] An E. coli <u>catalase</u> negative host strain CAT500 was infected with a phage solution containing sheared pieces of DNA from <u>Alcaligenes (Deleva)</u> aquanwrinus in pBluescript plasmid and plated on agar containing LB with ampicillin (100 .mu.g/mL), methicillin (80 .mu.g/mL) and kanamycin (100 .mu.g/mL) according to the method of Hay and Short (Hay, B. and Short, J., J. Strategies, 5:16, 1992). The resulting colonies were picked with sterile toothpicks and used to singly inoculate each of the wells of 96-well microliter plates. The wells contained 250 .mu.L of SOB media with 100 .mu.g/mL ampicillin, 80 .mu.g/mL methicillin, and (SOB Amp/Meth/Kan). The cells were grown overnight at 37.degree. C. without shaking. This constituted generation of the "SourceGeneBank"; each well of the Source GeneBank thus contained a stock culture of E. coli cells, each of which contained a pBluescript plasmid with a unique DNA insert. Same protocol was adapted for screening <u>catalase</u> from Microscilla furvescens.

Detail Description Paragraph - DETX (86):
[0091] <u>Microscilla furvescens catalase</u>: (.sub.PQET vector)

6/30/04, EAST Version: 2.0.0.29

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20030036187 A1

TITLE:

Novel bacteria strain having heavy oil degrading ability, bacteria mixture, heavy oil degrading bacteria nuturing composition, formulation containing that composition, method of treating oil components, and building and civil engineering materials containing

substance treated by that method

PUBLICATION-DATE:

February 20, 2003

INVENTOR-INFORMATION:

NAME

CITY

STATE COUNTRY RULE-47

Fujita, Tokio

Nara-shi

APPL-NO:

09/518814

DATE FILED: March 3, 2000

CONTINUED PROSECUTION APPLICATION: This is a publication of a continued prosecution application (CPA) filed under 37 CFR 1.53(d).

FOREIGN-APPL-PRIORITY-DATA:

COUNTRY APPL-NO

DOC-ID

APPL-DATE

JΡ 11-87162 1999JP-11-87162

March 29, 1999

US-CL-CURRENT: 435/281, 435/252.1, 435/262

ABSTRACT:

A bacteria strain FERMBP-7046 belonging to the genus Acinetobacter, a strain FERMBP-7049 belonging to the genus Acinetobacter, a strain FERMBP-7047 belonging to the genus Pseudomonas, and a strain FERMBP-7048 belonging to the genus Alcaligenes are caused to act on an object of treatment, either individually or in a bacteria mixture including at least one of the foregoing strains. Thus it is possible to provide heavy oil degrading bacteria and a heavy oil degrading bacteria mixture which are inexpensively prepared, which simplify degradation and removal operations, and which can be stored and shipped simply, and to provide a nurturing composition for such bacteria, a method of degrading heavy oil using such bacteria, and building and civil engineering materials containing a substance obtained by heavy oil degradation treatment.

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Detail Description Paragraph - DETX (35):

[0064] The strain TFBOL-3 was a Gram negative short bacillus having maneuverability, which did not develop under anaerobic conditions, showed positive in both the catalase reaction and oxidase reaction, and did not generate acid from glucose. Based on these results, the TFBOL-3 strain was identified as bacteria of the genus Alcaligenes.

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
Lj.	9051	catalase\$1	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
L2	2967	alcaligenes or deleya or aquamarinus or microscilla or furvescens	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
L3	47	1 same 2	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:02
<u>(4)</u>	77	1 near5 (muta\$10 or variant\$1)	US-PGPUB; USPAT	OR	OFF	2004/06/30 12:16

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20040109875 A1

TITLE:

Pro-apoptotic bacterial vaccines to enhance cellular

immune responses

PUBLICATION-DATE:

June 10, 2004

INVENTOR-INFORMATION:

NAME

STATE COUNTRY RULE-47

Kernodle, Douglas S.

Brentwood

US ΤN

Bochan, Markian R

Nashville

TN US

APPL-NO:

10/467644

DATE FILED: January 20, 2004

PCT-DATA:

APPL-NO: PCT/US02/03451 DATE-FILED: Feb 7, 2002

PUB-NO: PUB-DATE: 371-DATE: 102(E)-DATE:

US-CL-CURRENT: 424/200.1, 435/252.3

ABSTRACT:

Whole-cell vaccines and methods for their use in producing protective immune responses in vertebrate hosts subsequently exposed to pathogenic bacteria. The present invention involves a method of enhancing antigen presentation by intracellular bacteria in a manner that improves vaccine efficacy. After identifying an enzyme that has an anti-apoptotic effect upon host cells infected by an intracellular microbe, the activity of the enzyme is reduced, thereby modifying the microbe so that it increases immunogenicity. Also, the present invention provides a method of incrementally modifying enzyme activity to produce incrementally attenuated mutants of the microbe from which an effective vaccine candidate can be selected.

[0001] This application claims priority to U.S. provisional applications Serial No. 60/322,989 filed on Sep. 18, 2001, and Serial No. 60/267,328, filed Feb. 7, 2001. The 60/322,989 and 60/267,328 provisional patent applications are herein incorporated by this reference in their entirety.

 KWIC	
 NVVIC	

Detail Description Paragraph - DETX (243):

[0278] Because the host background of M. tuberculosis is different from E. coli, reversion analysis is performed directly with the H37Rv mutant. The goal is to monitor the in vivo phenotype in a manner that permits detection of any

enhanced fitness of the H37Rv strain bearing a soda mutant, including any compensatory changes in the production of other enzymes. For example, some catalase (katG) negative mutants of M. tuberculosis exhibit increased production of alkyl hydroperoxidase C (ahpC) resulting in some restoration of in vivo pathogenicity (Wilson, de Lisle, Marcinkeviciene, Blanchard, and Collins, 1998). By analogy, in addition to the potential for reversions or compensatory mutations in the soda allele, there could be compensatory overproduction of the zinc-copper SOD encoded by sodC that might partially restore virulence. Because of the unique secreted nature of the Fe-SOD, it is possible that even high-level non-secreted sodC overproduction might not completely complement the sodA mutants.

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20040072218 A1

TITLE:

Methods and kits for identifying scavengers of reactive

oxygen species (ros)

PUBLICATION-DATE:

April 15, 2004

INVENTOR-INFORMATION:

NAME

STATE COUNTRY RULE-47

Quan Pan, Shen

Singapore

SG

APPL-NO:

10/467104

DATE FILED: August 4, 2003

PCT-DATA:

APPL-NO: PCT/SG02/00018 DATE-FILED: Feb 5, 2002

PUB-NO: PUB-DATE: 371-DATE: 102(E)-DATE:

US-CL-CURRENT: 435/6

ABSTRACT:

This invention relates to methods and kits for determining the level of H.sub.2O.sub.2 inside a cell, and for determining whether a test compound has ability to scavenge a reactive oxygen species (ROS). The methods and diagnostic kits of this invention employ a cell containing a promoter which is inducible by an ROS, such as the H.sub.2O.sub.2-inducible KatA promoter of Agrobacterium tumefacines; the promoter is used to drive expression of a reporter gene. The expression level of the reporter gene correlates with the level of ROS, specifically H.sub.2O.sub.2, inside the cell. When the cell is exposed to a test compound, either intra-cellularly or extra-cellularly, if the level of ROS-inducible expression of the reporter is reduced, then this indicates that the test compound is a scavenger of the ROS. The methods of this invention may also be used to select for new or improved ROS scavengers by expressing a library of test scavengers in cells which express a reporter from a ROS-inducible promoter and selecting for those cells whose level of ROS-inducible expression of the reporter is reduced. The cells which express a reporter from a ROS-inducible promoter may have a modified genetic background to reduce their naturally occurring ROS-scavenging ability.

REFERENCE TO RELATED APPLICATIONS

[0001] This application claims the benefit of U.S. Provisional Application No. 60/266,657, filed Feb. 5, 2001, the content of which is herein incorporated by reference.

	KWI	<u> </u>
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Detail Description Paragraph - DETX (76):

[0113] The choice of bacterial strain to express the particular RIP-reporter construct and thus useful in the methods and kits of this invention is only limited by the strain's ability to produce the functional reporter and its inability to synthesize the reporter in its untransformed state. Most preferably, the strain used should be defective in genes which endogenously remove ROS intracellularly. Such genes include those encoding catalase, superoxide dismutase, alkyl hydroperoxidase, and glutathione reductase. For example, where an H.sub.2O.sub.2-inducible promoter is used, it is preferred that the endogenous catalase genes be knocked out or mutated in the cells so that the cells lose or have decreased capacity to break down H.sub.2O.sub.2 endogenously.

Detail Description Paragraph - DETX (112):

[0145] To determine whether the katA gene encodes a functional <u>catalase</u>, the <u>catalase isozyme patterns were analyzed for the mutant</u>, parent strain and complemented strain by using a catalase activity staining procedure. As shown in FIG. 2A, both the parent strain A348 and the complemented strain AG6 (pXQ9) had three distinct catalase activity bands (I, II and III), whereas the mutant AG6 had only one band (I). This demonstrated that the transposon insertion at the katA gene in AG6 knocked out two catalase activity bands.

Detail Description Paragraph - DETX (151):

[0168] Site-directed <u>mutagenesis was conducted to inactivate the catalase</u> activity. Computer analysis revealed that Arg 94 and His 98 in the conservative motif of KatA might be crucial for the catalase activity. Previous studies have indicated that the corresponding residues Arg 102 and His 106 of the E. coli homolog HPI are important for the catalase activity (Hillar et al., 2000). His 98 was changed to Asp [designated as KatA (98H/D)]; Arg 94 was changed to Gln and His 98 was changed to Asp [designated as KatA (94R/Q) (98H/D)]. The resulting plasmids were named pXQ26 and pXQ27, respectively.

Detail Description Paragraph - DETX (152):

[0169] As shown in FIG. 8, both the wild-type strain A348 and the complemented strain AG6 (pXQ23) had the KatA <u>catalase activity bands</u>, <u>whereas they were missing in the mutant</u> AG6, AG6 (pXQ26) and AG6 (pXQ27). This suggests that alteration of His 98 or both Arg 94 and His 98 in the conservative motif of KatA abolished the KatA catalase activity detected by the staining procedure. Western blot analysis was performed to check the stability of the mutant proteins. As shown in FIG. 6A, AG6 (pXQ26) and AG6 (pXQ27) produced the same size of KatA proteins as the wild type, and a high level of the point mutant proteins accumulated in the cells. As shown in FIG. 7, these two point mutant proteins could slightly repress the katA-gfp expression. The GFP expression was virtually undetectable in AG6 (pXQ23) (lane 4), but it was reduced in AG6 (pXQ26) and AG6 (pXQ27) (lanes 5 and 6), as compared with the strains expressing no or truncated protein KatA (lanes 7, 8, 9 and 10).

Detail Description Paragraph - DETX (154):

[0171] As shown in Table 2, the <u>catalase activity in the bacteria containing the mutant</u> protein, KatA.DELTA.86, KatA (94R/Q) (98H/D) or KatA.DELTA.50 was slightly higher than that in AG6, but not at a statistically significant level. This suggests that these mutant KatA proteins did not possess any significant catalase activity. The activities in whole cells for those bacteria were apparently due to the catalase other than KatA, since AG6 (which lacks katA) possessed catalase activity (FIG. 8 and Table 2). The catalase activity in AG6 (pXQ26) was statistically higher than that of AG6, suggesting that KatA (98H/D) possessed a low level of catalase activity. This low activity presumably has contributed to the low repression of katA-gfp (FIG. 7).

Detail Description Paragraph - DETX (168):

[0179] Two mutant KatA proteins truncated at the C-terminus exhibited a very low level of accumulation in the cells and no significant <u>catalase activity</u>; <u>neither mutant</u> repressed katA-gfp expression at any significant level (FIGS. 7 and 8; Table 2). These indicate that the katA-gfp expression levels can reflect the intracellular H.sub.2O.sub.2 scavenging capacity of the cells. If the cells are introduced with molecules, large or small, that can scavenge the intracellular H.sub.2O.sub.2 levels, the katA-gfp expression will be repressed. Therefore, measuring the katA-gfp expression can monitor the intracellular H.sub.2O.sub.2 scavenging capacity of a molecule that is introduced into the cells.

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20040067500 A1

TITLE:

Compositions and methods relating to the peroxisomal

proliferator activated receptor-alpha mediated pathway

PUBLICATION-DATE:

April 8, 2004

INVENTOR-INFORMATION:

NAME

STATE

COUNTRY RULE-47 US

Gould-Rothberg, Bonnie Sundseth, Scott

New Haven Durham

CT NC US

Gottschalk, William

Chapel Hill

NC US

APPL-NO:

10/391706

DATE FILED: March 18, 2003

RELATED-US-APPL-DATA:

child 10391706 A1 20030318

parent continuation-of 09440315 19991112 US GRANTED

parent-patent 6551812 US

non-provisional-of-provisional 60108293 19981113 US

non-provisional-of-provisional 60126465 19990326 US

US-CL-CURRENT: 435/6, 435/7.2

ABSTRACT:

The present invention describes polynucleotides and polypeptides associated with PPAR.alpha.-mediated pathways that are useful as therapeutic compositions in method for the treatment of peroxisomal disorders. These polynucleotides and polypeptides were identified through the use of differential gene expression analysis. In particular, the present invention discloses eleven novel gene fragments, and numerous single nucleotide polymorphisms, located in previously disclosed genes, all of which have been discovered to be associated with PPAR.alpha.-mediated pathways.

RELATED APPLICATIONS

[0001] This application is a continuation of U.S. Ser. No. 09/440,315, filed Nov. 12, 1999, which claims priority to U.S. S. No. 60/108,293, filed Nov. 13, 1998, and No. 60/126,465, filed Mar. 26, 1999, each of which are incorporated herein by reference in their entirety.

----- KWIC -----

Detail Description Paragraph - DETX (18): [0032] Active PPAR.alpha. ligands induce peroxisome proliferation along with an increase in peroxisomal fatty acid .alpha. oxidation. Peroxisomal beta.-oxidation is thus a primary target of the PPAR.alpha.response. Genes for all three steps of this biochemical pathway have upregulated transcription (See, e.g., Marcus, et al., 1993, Proc Natl Acad Sci USA 90:5723-5727). The differential gene expression analyses herein confirm two of these steps: acyl-CoA oxidase and the enoyl-CoA hydratase/2-hydroxyl-CoA dehydrogenase. Additionally, six more genes encoding PPAR.alpha.L-responsive genes described herein are involved in the peroxisomal .beta.-oxidation cascade: (i) Very long chain acyl-CoA synthase; (ii) Camitine octanoyl transferase; (iii) Acyl-CoA hydrolase; (iv) acyl-CoA thioesterase; (v) Catalase; and (vi) acyl-CoA oxidase variant. Very long chain acyl-CoA synthase is a peroxisome-specific acyl-CoA synthase responsible for preparing very long chain fatty acids for .beta.-oxidation. Carnitine octanoyl transferase translocates medium chain fatty acids across the peroxisomal membrane for subsequent degradation. Acyl-CoA hydrolase and acyl-CoA thioesterase are two genes responsible for modifications of acyl-CoAs and their release from fatty acid oxidation. Catalase is the enzyme responsible for neutralizing peroxide radicals, and was also upregulated. Another enzyme identified herein may be a novel acyl-CoA oxidase variant. This gene is of particular interest as it could indicate the presence of several acyl-CoA oxidases that might function in parallel during peroxisomal .beta.-oxidation.

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20040047852 A1

TITLE:

Method of treating cancer

PUBLICATION-DATE:

March 11, 2004

INVENTOR-INFORMATION:

NAME

CITY

STATE COUNTRY RULE-47

Kennedy, Thomas Preston

Charlotte

NC US

APPL-NO:

10/258308

DATE FILED: September 3, 2003

PCT-DATA:

APPL-NO: PCT/US01/40237 DATE-FILED: Mar 2, 2001

PUB-NO: PUB-DATE: 371-DATE: 102(E)-DATE:

US-CL-CURRENT: 424/94.4, 514/492, 514/562

ABSTRACT:

Methods for treating cancer are provided including administering a patient needing treatment a therapeutically effective amount of one or more antioxidants selected from the group of catalase, N-acetylcysteine, glutathione peroxidase, salen-transition metal complexes, dicumarol, and derivatives thereof.

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Detail Description Paragraph - DETX (15):

[0041] Various forms of catalases have been identified and isolated from organisms including animals, plants, fungi and bacteria. Typically, catalase has a molecular weight of about 240 Kilodaltons. Any forms of catalase can be used in the present invention so long as it has catalytic activity of decomposing hydrogen peroxide in mammalian cells. Catalases isolated from animal livers (bovine hepatocatalase) and kidneys as well as bacteria (e.g., Micrococcus Lysodeikticus) and fungi (e.g., Aspergillus Niger) are commercially available and can all be used in the present invention. Catalase from other sources, e.g., produced by genetic engineering, can also be used. In addition, various modified forms or derivatives of catalase can be used. For example, muteins, i.e., mutant forms of catalase containing fragments of catalase or having substituent amino acids in the polypeptide chain, can be useful. Catalase conjugated with polyethylene glycol is especially useful in the present invention because of its reduced immunogenicity and increased stability.

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20040034100 A1

TITLE:

Therapeutic malonic acid/acetic acid C60 tri-adducts of

IL

buckminsterfullerene and methods related thereto

PUBLICATION-DATE:

February 19, 2004

INVENTOR-INFORMATION:

NAME CITY

COUNTRY RULE-47 STATE

Dugan, Laura L.

St.Louis

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Lovett, Eva G.

University City

MO US

Quick, Kevin L. Hardt, Joshua I. Florissant Belleville

MO US US

APPL-NO:

10/373425

DATE FILED: February 24, 2003

RELATED-US-APPL-DATA:

child 10373425 A1 20030224

parent continuation-in-part-of 10083283 20020223 US PENDING

US-CL-CURRENT: 514/569, 562/488

ABSTRACT:

Disclosed and claimed herein are e.e.e malonic acid/acetic acid tri-adduct of buckminsterfullerene of the general formula C.sub.60R.sub.3, wherein each R is independently selected from groups of the formula -- CR.sup.1R.sup.2 wherein each R.sup.1 and R.sup.2 is independently selected from the group consisting of --H and --COOH, provided, however, that at least one of the R.sup.1's and R.sup.2's is a hydrogen. Processes for preparing and uses of the same for treating neuronal injury and for life-extension are also disclosed and claimed herein.

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Summary of Invention Paragraph - BSTX (9):

[0007] For example, the genetic analysis of C.elegans has revealed several genes involved in lifespan determination. Mutations in Daf-2 (the insulin receptor) and Clk-1 ("Clock 1", a gene affecting many aspects of developmental and behavioral timing) have been shown to extend the lifespan of adults. However, Clk-1 mutants have a higher mortality rate in early life. At later stages of development, the Clk-1 mutants show an increase in longevity, perhaps by selecting for long-lived individuals in early life. The Clk-1 longevity phenotype is abolished by mutations in the gene encoding catalase, which is involved in superoxide/free radical metabolism. Additionally, elimination of coenzyme Q in C. elegans diet has been shown to extend lifespan.

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20040023283 A1

TITLE:

90 human secreted proteins

PUBLICATION-DATE: February 5, 2004

INVENTOR-INFORMATION:

	D (11 Q14.			
NAME	CITY	STATE	COUNTRY	RULE-47
Ruben, Steven M.	Brookeville	MD	US	
Soppet, Daniel R.	Centreville	VA	US	
Ebner, Reinhard	Gaithersburg	MD	US	
Olsen, Henrik S.	Gaithersburg	MD	US	
Young, Paul E.	Gaithersburg	MD	US	
Greene, John M.	Gaithersburg	MD	US	
Ferrie, Ann M.	Painted Post	NY	US	
Yu, Guo-Liang	Berkeley	CA	US	
Ni, Jian	Germantown	MD	US	
Rosen, Craig A.	Laytonsville	MD	US	
Brewer, Laurie	St. Paul	MN	US	
Janat, Fouad	Westerly	RI	US	
Birse, Charles E.	North Potomac	MD	US	

APPL-NO: 10/621363

DATE FILED: July 18, 2003

RELATED-US-APPL-DATA:

child 10621363 A1 20030718

parent continuation-of 09969730 20011004 US PENDING

child 10621363 A1 20030718

parent continuation-in-part-of 09774639 20010201 US PENDING

child 09774639 20010201 US

parent continuation-of 09244112 19990204 US ABANDONED

child 09244112 19990204 US

parent continuation-in-part-of PCT/US98/16235 19980804 US PENDING

non-provisional-of-provisional 60238291 20001006 US

non-provisional-of-provisional 60055386 19970805 US

non-provisional-of-provisional 60054807 19970805 US

non-provisional-of-provisional 60055312 19970805 US

non-provisional-of-provisional 60055309 19970805 US

6/30/04, EAST Version: 2.0.0.29

non-provisional-of-provisional 60054798 19970805 US non-provisional-of-provisional 60055310 19970805 US non-provisional-of-provisional 60054806 19970805 US non-provisional-of-provisional 60054809 19970805 US non-provisional-of-provisional 60054804 19970805 US non-provisional-of-provisional 60054803 19970805 US non-provisional-of-provisional 60054808 19970805 US non-provisional-of-provisional 60055311 19970805 US non-provisional-of-provisional 60055986 19970818 US non-provisional-of-provisional 60055970 19970818 US non-provisional-of-provisional 60056563 19970819 US non-provisional-of-provisional 60056557 19970819 US non-provisional-of-provisional 60056731 19970819 US non-provisional-of-provisional 60056365 19970819 US non-provisional-of-provisional 60056367 19970819 US non-provisional-of-provisional 60056370 19970819 US non-provisional-of-provisional 60056364 19970819 US non-provisional-of-provisional 60056366 19970819 US non-provisional-of-provisional 60056732 19970819 US non-provisional-of-provisional 60056371 19970819 US

US-CL-CURRENT: 435/6, 435/183 , 435/320.1 , 435/325 , 435/69.1 , 530/350 , 536/23.2

ABSTRACT:

The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CROSS REFERENCE TO RELATED APPLICATIONS

[0001] This application is a continuation of U.S. application Ser. No. 09/969,730, filed on Oct. 4, 2001, which claims benefit under 35 U.S.C. .sctn. 119(e) of U.S. Provisional Application No. 60/238,291, filed Oct. 6, 2000; U.S. application Ser. No. 09/969,730 is also a continuation-in-part of

U.S. application Ser. No. 09/774,639, filed on Feb. 1, 2001, which is a continuation of U.S. application Ser. No. 09/244,112, filed on Feb. 4, 1999 (abandoned), which is a continuation-in-part of International Application No. PCT/US98/16235, filed on Aug. 4, 1998, which claims benefit under 35 U.S.C. .sctn. 119(e) of U.S. Provisional Application Nos. 60/055,386, 60/054,807, 60/055,312, 60/055,309, 60/054,798, 60/055,310, 60/054,806, 60/054,809, 60/054,804, 60/054,803, 60/054,808, and 60/055,311, all of which were filed on Aug. 5, 1997, 60/055,986 and 60/055,970, both of which were filed on Aug. 18, 1997, and 60/056,563, 60/056,557, 60/056,731, 60/056,365, 60/056,367, 60/056,370, 60/056,364, 60/056,366, 60/056,732, and 60/056,371, all of which were filed on Aug. 19, 1997. U.S. application Ser. Nos. 09/969,730, 09/774,639, and 09/244,112; International Application No. PCT/US98/16235; and U.S. Provisional Application Nos. 60/238,291, 60/055,386, 60/054,807, 60/055,312, 60/055,309, 60/054,798, 60/055,310, 60/054,806, 60/054,809, 60/054,804, 60/054,803, 60/054,808, 60/055,311, 60/055,986, 60/055,970, 60/056,563, 60/056,557, 60/056,731, 60/056,365, 60/056,367, 60/056,370, 60/056,364, 60/056,366, 60/056,732, and 60/056,371 are all hereby incorporated by reference in their entirety.

----- KWIC -----

Summary of Invention Paragraph - BSTX (494):

[0491] The tissue distribution in neutrophils indicates that polynucleotides and polypeptides corresponding to this gene would be useful for the diagnosis, detection, prevention and/or treatment of a variety of immune system disorders. Expression of this gene product indicates that polynucleotides and/or polypeptides of the invention may play a role in regulating the proliferation; survival; differentiation; and/or activation of potentially all hematopoietic cell lineages, including blood stem cells. Polynucleotides and/or polypeptides corresponding to this gene may be involved in the regulation of cytokine production, antigen presentation, or other processes that may also indicate a usefulness in the treatment of cancer (e.g., by boosting immune responses). Since the gene is expressed in cells of lymphoid origin, the natural gene product may be involved in immune functions. Therefore polynucleotides and/or polypeptides corresponding to this gene may be also used as an agent for immunological disorders including arthritis, asthma, immune deficiency diseases such as AIDS, leukemia, rheumatoid arthritis, inflammatory bowel disease, sepsis, acne, and psoriasis, and tissues. In addition, this gene product may have commercial utility in the expansion of stem cells and committed progenitors of various blood lineages, and in the differentiation and/or proliferation of various cell types. Alternatively, the homology to catalase indicates that polynucleotides and polypeptides corresponding to this gene would be useful for the study, detection, treatment, and/or prevention of a variety of metabolic disorders. As elevated levels of peroxide in cells and tissues leads to oxidative damage, largely through the generation of oxide free-radicals, mutations within the catalase gene may lead to the accumulation of cellular mutations over time and could predispose an individual to cancer or other disorder and disease. Protein, as well as, antibodies directed against the protein may show utility as a tumor marker and/or immunotherapy targets for the above listed tissues.

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20030228673 A1

TITLE:

Novel glucose 6-oxidases

PUBLICATION-DATE:

December 11, 2003

INVENTOR-INFORMATION:

NAME

STATE **COUNTRY RULE-47**

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APPL-NO:

10/375909

DATE FILED: February 27, 2003

RELATED-US-APPL-DATA:

non-provisional-of-provisional 60359878 20020227 US

non-provisional-of-provisional 60400417 20020801 US

US-CL-CURRENT: 435/189, 435/254.7, 435/320.1, 435/69.1, 536/23.2, 702/19

ABSTRACT:

Glucose oxidase enzymes are provided, including novel variants of galactose oxidase enzymes. The polynucleotides that encode these novel variants can be expressed in recombinant host cell expression systems. The novel variant oxidase enzymes are capable of oxidizing compounds towards which wild-type galactose oxidase (e.g. D-galactose: oxygen 6-oxidoreductase, GAO; EC 1.1.3.9) has little or no activity. Preferred galactose oxidase variants are those which that have improved capability to oxidize secondary alcohols and/or D-glucose relative to the wild-type enzyme.

[0001] This application claims priority under 35 U.S.C. .sctn.119(e) to U.S. provisional patent applications No. 60/359,878, filed Feb. 27, 2002, and No. 60/400,417, filed Aug. 1, 2002, each of which is hereby incorporated by reference in its entirety.

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	 K WIII	

Detail Description Paragraph - DETX (121):

[0134] Oxidation reaction. Oxidation of methyl-.beta.-D-galacto-pyranoside with Sigma GAO was performed in 5 ml NaPi (50 mM, pH 7.0) with 300 mM substrate, 95 U of Sigma GAO, 700 U catalase (Sigma) and 0.5 mM copper ion. Oxidation of methyl-.beta.-D-gluco-pyranoside with M-RQW was performed in 2 ml of NaPi (50 mM, pH 7.0) with 200 mM substrate, 1.8 U mutant, 1700 U catalase and 0.5 mM copper ion. Both reactions were performed at room temperature with vigorous stirring. 2-Butanol oxidation by M-RQW was performed at room temperature in 100 .mu.l NaPi (50 mM, pH 7.0) containing 50 mM substrate, 0.5

mM copper ion, 0.9 U $\underline{\text{mutant GAO}}$ and 150 U catalase.

PGPUB-FILING-TYPE:

new

DOCUMENT-IDENTIFIER: US 20030228581 A1

TITLE:

Diagnosis and treatment for late onset

neurodegenerative disorder

PUBLICATION-DATE:

December 11, 2003

INVENTOR-INFORMATION:

NAME

CITY

STATE **COUNTRY RULE-47**

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ME US

Ackerman, Susan L.

Bar Harbor

ME US

APPL-NO:

10/ 164295

DATE FILED: June 6, 2002

US-CL-CURRENT: 435/6

ABSTRACT:

Disclosed is a method for diagnosing the molecular basis for a late-onset neurodegenerative disorder in a mammal. In this method, a sample from the mammal is provided, the sample containing a nucleic acid sequence encoding the AIF. The sequence of nucleic acids in the nucleic acid sequence is determined by conventional techniques. This determined sequence of nucleic acids is then compared to a nucleic acid sequence encoding the wild-type AIF protein. Any difference between the sequence of nucleic acids determined from the two samples represents a candidate mutation. The candidate mutation is then further analyzed for an association with a decrease in expression of functional AIF protein. Such an association confirms a molecular basis for the late-onset neurodegenerative disorder in the mammal. Related methods and compositions are also disclosed.

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Detail Description Paragraph - DETX (23):

[0036] To examine if a reduction in AIF leads to conditions of oxidative stress, antioxidant enzyme levels, lipid peroxidation and DNA oxidative damage in Hq and wild type mice were examined. Due to the similarity of the oxidoreductase moiety of AIF to bacterial hydrogen peroxide scavengers, it was hypothesized that changes in catalase and glutathione levels would occur in Hq mutant mice. Catalase is the major scavenger of hydrogen peroxide in cellular systems (reviewed in Deisseroth et al., Physiol. Rev. 50: 319-375 (1970)), while glutathione is an essential electron donor for the reduction of hydroperoxides (Baillie et al., Acc. Chem. Res. 24: 264-270 (1991)). In agreement with this, catalase activity is increased in the cerebella of Ho mutant mice at both 1 and 3 months of age compared to wild type levels. However, no differences were noted in the remainder of the brain at either one or three months (p>0.05). As with catalase, total glutathione levels were increased in the cerebella of Hg mutant mice at both one and three months of age compared to wild type levels. No differences were observed in total glutathione levels in the remainder of the brain at either one or three months (p>0.05). Western blot analysis of cerebellar extracts of one and three

month old Hg mutant and wild type mice showed increases in catalase expression consistent with the increased catalase activity, while no differences in either SOD1 or SOD2 levels were observed at either age.

US-PAT-NO:

6709815

DOCUMENT-IDENTIFIER: US 6709815 B1

TITLE:

Target-dependent reactions using structure-bridging

oligonucleotides

DATE-ISSUED:

March 23, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CO	DE COUNTRY
Dong; Fang	Madison	WI	N/A	N/A
Lyamichev; Victor I.	Madison	IW	N/A	N/A
Prudent; James R.	Madison	WI	N/A	N/A
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Neri; Bruce P.	Madison	WI	N/A	N/A
Brow; Mary Ann D.	Madison	WI	N/A	N/A
Anderson; Todd A.	Madison	WI	N/A	N/A
Dahlberg; James E.	Madison	WI	N/A	N/A

APPL-NO:

09/402618

DATE FILED: July 18, 2000

PARENT-CASE:

This application is a 371 of PCT/US98/03194 filed May 5, 1998 which is a CIP of Ser. No. 08/851,588 filed May. 5, 1997, now U.S. Pat. No. 6,214,545, and a CIP of Ser. No. 08/934,097 filed Sep. 19, 1997, now U.S. Pat. No. 6,210,880, and a CIP of Ser. No. 08/034,205 filed Mar. 3, 1998 and now U.S. Pat. No. 6,194,149.

US-CL-CURRENT: 435/6, 536/23.1, 536/24.3

ABSTRACT:

The present invention relates to methods and compns. for treating nucleic acids, and in particular, methods and compns. for the detection and characterization of nucleic acid sequences and sequence changes. The invention provides methods for examg. the conformations assumed by single strands of nucleic acid, forming the basis of novel methods of detection of specific nucleic acid sequences. The present invention contemplates use of novel detection methods for, among other uses, clinical diagnostic purposes, including but not limited to the detection and identification of pathogenic organisms. Examples are presented for the analysis of Mycobacterium tuberculosis and hepatitis C virus genes.

47 Claims, 65 Drawing figures

Exemplary Claim Number:

Number of Drawing Sheets: 65

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Other Reference Publication - OREF (12):

Cockerill, III et al., "Rapid Identification of a Point <u>Mutation of the Mycobacterium tuberculosis Catalase</u>-Peroxidase (katG) Gene Associated with Isoniazid Resistance," J. Infect. Dis. 171:240-245 [1995].

US-PAT-NO:

6689760

DOCUMENT-IDENTIFIER: US 6689760 B1

TITLE:

Anti-mycobacterial compositions

DATE-ISSUED:

February 10, 2004

INVENTOR-INFORMATION:

NAME

CITY

STATE ZIP CODE COUNTRY

Yatvin; Milton B.

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N/A N/A

Pederson; Richard L.

San Gabriel

OR

CA N/A N/A

APPL-NO:

09/613409

DATE FILED: July 10, 2000

US-CL-CURRENT: 514/45, 514/47, 536/26.24

ABSTRACT:

This invention provides compositions of matter, pharmaceutical compounds, methods of synthesizing such compounds and methods for using such compounds to treat animals infected with a pathogenic mycobacterium. The invention specifically provides compositions and pharmaceutical compositions thereof for the treatment of tuberculosis and other Mycobacterium-caused diseases.

17 Claims, 13 Drawing figures

Exemplary Claim Number:

1,11,14,15

Number of Drawing Sheets: 13

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Brief Summary Text - BSTX (13):

Each of the compounds disclosed herein is an analog for nicotinamide adenine dinucleotide, a cellular component that mediates transfer of electrons in a number of cellular systems (including glycolysis, mitochondrial oxidative phosphorylation, fatty acid synthesis and breakdown, and other synthetic and metabolic pathways). It is known in the art that isoniazid, the traditional drug of choice for treating tuberculosis, is activated by a M. tuberculosis produced catalase/peroxidase (as shown in FIG. 1); Quemard et al., 1996, J. Amer. Chem. Soc. 118: 1561; Sacchettini & Blanchard, 1996, Res. Microbiol. 147: 36; Zabinski & Blanchard, 1997, J. Amer. Chem. Soc. 119: 2331) to form an adduct with NAD (the resulting activated form of isoniazid is termed isoniazid-NAD analogue (INA); Rozwarski et al., 1998, Science 279: 98-102). A major route for isoniazid resistance to M. tuberculosis is mutation or inactivation of the catalase/peroxidase that converts isoniazid to INA. suggesting that mycobacteria may be less likely to develop resistance to INA that to isoniazid itself.

Detailed Description Text - DETX (5):

A limitation of such therapy, however, is the development of drug resistance by the mycobacterium. This phenotype is frequently expressed by as loss-of-function mutation involving a mycobacterially-encoded

<u>catalase</u>/-peroxidase that prevents activation of isoniazid to the active adduct shown in FIG. 1. Without mycobacterial-mediated activation, the drug loses its anti-mycobacterial properties.

US-PAT-NO:

6673616

DOCUMENT-IDENTIFIER: US 6673616 B1

TITLE:

Methods and compositions for characterizing nucleic

acids

DATE-ISSUED:

January 6, 2004

INVENTOR-INFORMATION:

NAME

CITY

STATE ZIP CODE COUNTRY

Dahlberg; James E. Brow; Mary Ann D.

Madison Madison

N/A N/A N/A

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Madison

WI N/A WI N/A N/A

WI

APPL-NO:

09/655378

DATE FILED: September 5, 2000

PARENT-CASE:

This is a Continuation of U.S. patent application Ser. No. 08/520,946, filed Aug. 30, 1995, now U.S. Pat. No. 6,372,424, which is a Continuation-in-Part application of U.S. patent application Ser. No. 08/484,956, filed Jun. 7, 1995, now U.S. Pat. No. 5,843,654, issued Dec. 1, 1998, which is a Continuation-in-Part application of U.S. patent application Ser. No. 08/402,601, filed Mar. 9, 1995, now abandoned and the Continuation U.S. patent application Ser. No. 08/802,233, filed Feb. 19, 1997, now U.S. Pat. No. 5,888,780, issued Mar. 30, 1997, which is a Continuation-In-Part Application of application Ser. No. 08/337,164, filed Nov. 9, 1994 and the Continuation U.S. patent application Ser. No. 08/789,079, filed Feb. 6, 1997, now U.S. Pat. No. 5,719,028, issued Feb. 17. 1998, which is a Continuation-In-Part Application of application Ser. No. 08/254,359, filed Jun. 6, 1994, now U.S. Pat. No. 5,614,402, issued Mar. 25, 1997, which is a Continuation-In-Part Application of application Ser. No. 08/073,384, filed Jun. 4, 1993, now U.S. Pat. No. 5,541,311, issued Jun. 30, 1996, which is a Continuation-In-Part Application of application Ser. No. 07/986,330, filed Dec. 7, 1992, now abandoned. The present application also claims priority to U.S. patent application Ser. No. 08/471,066, filed Jun. 6, 1995, now U.S. Pat. No. 5,837,450, issued Nov. 17, 1998, which is a Divisional application of U.S. patent application Ser. No. 08/254,359, listed above, U.S. patent application Ser. No. 08/481,238, filed Jun. 6, 1995, now U.S. Pat. No. 5,795,763, issued Aug. 18, 1998, which is a Divisional application to U.S. patent application Ser. No. 07/986,330, listed above, and U.S. patent application Ser. No. 08/483,043, filed Jun. 6, 1995, now U.S. Pat. No. 5,691,142, issued Nov. 25, 1997, which is a Divisional application of U.S. patent application Ser. No. 07/986,330, listed above.

US-CL-CURRENT: 436/6, 536/23.1

ABSTRACT:

The present invention relates to means for cleaving a nucleic acid cleavage structure in a site-specific manner. Enzymes, including 5' nucleases and 3' exonucleases, are used to detect and identify nucleic acids derived from microorganisms. Methods are provided which allow for the detection and identification of bacterial and viral pathogens in a sample.

20 Claims, 151 Drawing figur	res	
Exemplary Claim Number:	1	
Number of Drawing Sheets:	124	
KWIC		
Other Reference Publication - OREF (109):		

US-PAT-NO:

6630582

DOCUMENT-IDENTIFIER: US 6630582 B1

TITLE:

Treatment and prevention of helicobacter infection

DATE-ISSUED:

October 7, 2003

INVENTOR-INFORMATION:

NAME CITY STATE ZIP CODE COUNTRY

Doidge: Christopher V. Lee: Adrian

Vincent Lane Cove

N/A N/A ΑU N/A N/A ΑU

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APPL-NO:

09/004014

DATE FILED: January 7, 1998

PARENT-CASE:

CROSS-REFERENCE TO RELATED APPLICATIONS

This application is a divisional of U.S. patent application Ser. No. 08/695987 filed Aug. 15, 1996 which is a continuation-in-part of International Patent Application No. PCT/AU 95/0335, dated Jun. 8, 1995, and designating the United States of America, the disclosure of which is incorporated herein by reference.

FOREIGN-APPL-PRIORITY-DATA:

COUNTRY

APPL-NO

APPL-DATE

ΑU

PM 6124

June 8, 1994

US-CL-CURRENT: 536/23.6, 424/234.1, 424/93.2, 435/192, 435/7.32

ABSTRACT:

An antigenic preparation for use in the treatment or prevention of Helicobacter infection in a mammalian host, comprises the catalase enzyme of Helicobacter bacteria, particularly the catalase enzyme of H. pylori or H. felis, or an immunogenic fragment thereof.

12 Claims, 0 Drawing figures

Exemplary Claim Number:

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Detailed Description Text - DETX (90):

6. Westblom, T. U., Phadnis, S., Langenberg, W., Yoneda, K., Madan, E. and Midkiff, B. R. (1992). Catalase negative mutants of Helicobacter pylori. European Journal of Clinical Microbiology and Infectious Diseases, 11:522-526.

Other Reference Publication - OREF (23):

Westblom, T.U., et al., "Catalase Negative Mutants of Helicobacter pylon," Eur. J. Clin. Microbiol. Infect. Dis. (Jun. 1992), vol. 11(6), pp. 522-526.